

**CLAIMS**

- 1) Method for assessing *in vitro* the predisposition of a subject to develop cardiovascular pathologies, characterized in that the identity of the nucleotide corresponding to position 436 of seq IDN1 (COX-2 gene PROMOTER is established on a sample of genomic DNA of said subject).  
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- 2) Method according to claim 1, where the genomic DNA is extracted from cells of such subject, derived from blood samples, saliva, biopsies, urine, human tissue.
- 3) Method according to claim 2, where said cardiovascular pathologies are caused by or associated with rupture of an atherosclerotic plaque.  
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- 4) Method according to claims 1-3, where such cardiovascular pathologies are coronaropathies, pathologies of carotid arteries, myocardial infarction, angina pectoris, acute coronary syndromes, myocardial revascularization by means of coronary by-pass or angioplasty, stroke, transient ischemic attack (TIA), peripheral arteriopathy, trombophylic syndromes.  
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- 5) Method according to claim 4, where such assessment is made by one of the following techniques: sequencing, endonuclease digestion with restriction enzymes, selective hybridization with oligonucleotides specific for polymorphism at position -765 of the human COX-2 gene promotor, methodology of single strand conformational polymorphism (SSCP), DGGE, Fluorescence assisted mismatch analysis (FAMA), heteroduplex analysis, Real Time PCR.  
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- 6) Method according to claim 5, wherein said assessment is made by endonuclease digestion with restriction enzymes.  
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- 7) Method according to claim 6, comprising the following steps:
  - extraction of genomic DNA from a biological sample of the subject,
  - amplification by means of Polymerase Chain Reaction with oligonucleotides or primers suitable for amplification of a DNA fragment comprising position -765,
  - enzymatic digestion of such amplified fragment with a restriction enzyme selected from: Fau I and Aci I
  - electrophoretic separation of the restriction mixture comprising the restriction fragments or of the undigested amplified fragment, or of both,  
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- analysis of the restriction profile generated after visualization of DNA.

8) Method according to claim 7, characterized in that the amplification is carried out with oligonucleotides having sequences at least partially identical to sequences ID NO 3 and ID NO 4 and the amplified fragment is digested with the restriction enzyme Fau I.

9) Method according to claim 8, characterized in that the amplification is carried out with oligonucleotides having sequence SEQ. ID NO 3 and 4.

10) Method according to claims 1-9, characterized in that the presence of a cytosine (C) at position 436 of SEQ IDN1, in at least one DNA allele of such subject, indicates a lower risk to predisposition to cardiovascular diseases than the risk associated to the presence of a guanosine (G) in position 436 on both alleles.

11) Kit in order to carry out the method according to claims 1-10.

12) Kit according to claim 11, characterized for comprising at least one of the following oligonucleotides: an oligonucleotide comprising at least 10 consecutive nucleotides of seq ID NO 3, an oligonucleotide comprising at least 10 consecutive nucleotides of seq ID NO 4 and optionally one restriction enzyme selected from: Fau I and Aci I.

13) Kit according to claim 12, comprising the oligonucleotide with sequence ID NO 3 and the oligonucleotide with sequence ID NO 4, the Fau I restriction enzyme and optionally one molecular weight DNA standard.

14) Use of the genotyping of nucleotide at position 436 of seq IDN1 (COX-2 gene promotor) for the preparation of a prognostic tests for a cardiovascular pathology selected from: coronaropathies, pathologies of carotid arteries, myocardial infarction, angina pectoris, acute coronary syndromes, myocardial revascularization by means of coronary by-pass or angioplasty, stroke, transient ischemic attack (TIA), peripheral arteriopathy, trombophilic syndromes.

15) Use of the genotypzation of nucleotide at position 436 of seq IDN1 (COX-2 gene promotor) to prepare diagnostic tests for the sensitivity to therapy with non steroidal anti-inflammatory drugs (FANS).